Relation between coronary risk and coronary mortality in women of the Renfrew and Paisley survey: comparison with men

CHRISTOPHER G. ISLES DAVID J. HOLE VICTOR M. HAWTHORNE
ANTHONY F. LEVER

Most epidemiological and intervention studies in patients with coronary artery disease have focused on men, the assumption being that such data can be extrapolated to women. However, there is little evidence to support this belief.

We have completed a fifteen-year follow-up of 15 399 adults, including 8262 women, who lived in Renfrew and Paisley and were aged 45–64 years when screened between 1972 and 1976. We identified 490 deaths from coronary heart disease (CHD) in women and 878 in men. Women were more likely to have high cholesterol, to be obese, and to come from lower social classes than men, but they smoked less and had similar blood pressures. The relative risk—top to bottom quintile (95% CI)—of cholesterol for coronary death after adjustment for all other risk markers was slightly greater in women (1·77 [1·45, 2·16]) than in men (1·56 [1·32, 1·85]), but absolute and attributable risk were lower. Thus, women in the top quintile for cholesterol had lower coronary mortality (6·1 deaths per thousand patient years) than men in the bottom quintile (6·8 deaths per thousand patient years). Moreover, it was estimated that there would have been only 103 (21%) fewer CHD deaths in women, yet 211 (24%) fewer in men, if mortality had been the same for women and men in the lowest quintiles of cholesterol. Trends showing similar relative risks in these women, but lower absolute and attributable risks than in men, were present for smoking, diastolic blood pressure, and social class. There was no relation between obesity and coronary death after adjustment for other risks.

Our results suggest that some other factors protect women against CHD. The potential for women to reduce their risk of CHD by changes in lifestyle may be less than for men.

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Introduction

Rates of coronary heart disease (CHD) increase with age in both sexes but are higher in men than in women.[1] Most population studies and intervention trials have focused on the relation between coronary risk and coronary disease, together with the effects of reducing cholesterol, in men. However, recommendations for prevention of coronary disease do not distinguish between men and women[2] on the grounds that what is good for men is likely to benefit women. This approach could only be justified if the absolute and attributable risks of CHD were similar, and if preventive measures were effective in both sexes. To examine risk further, we have analysed the relation of coronary heart disease with plasma cholesterol, cigarette smoking, diastolic blood pressure, body mass index, and social class in a general population of 15 399 middle-aged adults, including 8262 women, who were screened in Renfrew and Paisley between 1972 and 1976 and then followed up until the end of 1989.

Methods

The Renfrew and Paisley survey is a longitudinal health study of 15 399 subjects, including 8262 women, who were aged 45 to 64 years when first examined between 1972 and 1976. The subjects were taken as representative of an urban population in the west of Scotland. The survey was preceded by a census and each man and woman between the ages of 45 and 64 years was visited and given an invitation to attend; 79% of eligible subjects in Renfrew and 78% of those in Paisley did so. During the first two years of follow-up, those not attending had higher mortality than those who did attend. Thereafter, mortality rates were similar in attenders and non-attenders.

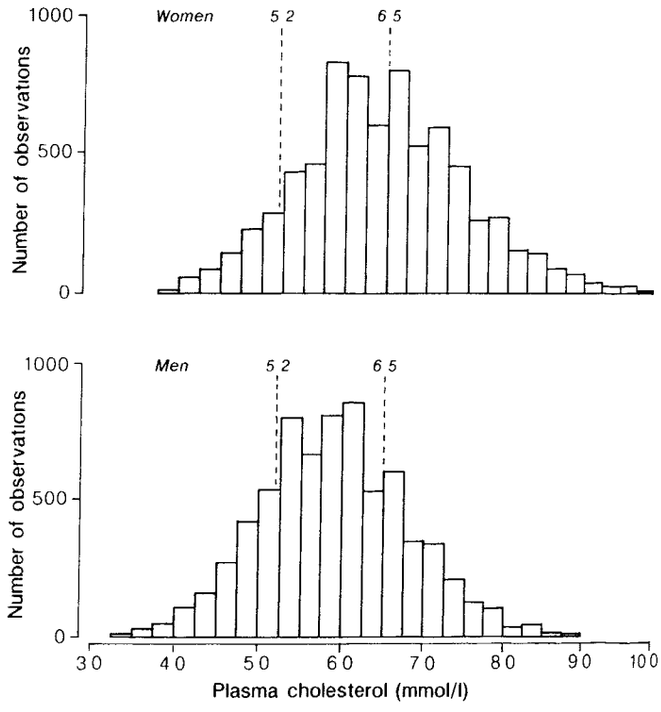

Fig 1—Distribution of plasma cholesterol among 8262 women and 7137 men aged 45–64 years.

Smoking habits were recorded by a standard questionnaire and measurements were made of height and weight (to give body mass index in kg/m²), blood pressure (London School of Hygiene sphygmomanometer, diastolic phase V), and non-fasting plasma cholesterol. Social class was determined by occupation, except for housewives and retired women whose husbands' or fathers' occupations were used instead in classification.

ADDRESSES: **Dumfries and Galloway Royal Infirmary, Dumfries, UK** (C. G Isles, FRCP); **Cancer Surveillance Unit, Ruchill Hospital, Glasgow, UK** (D. J. Hole, MSc); **Department of Epidemiology, University of Michigan, Michigan, USA** (Prof V. M. Hawthorne, MD); **and MRC Blood Pressure Unit, Western Infirmary, Glasgow, UK** (Prof A. F. Lever, FRCP) Correspondence to Dr C. G. Isles, Dumfries and Galloway Royal Infirmary, Dumfries DG1 4AP, UK.

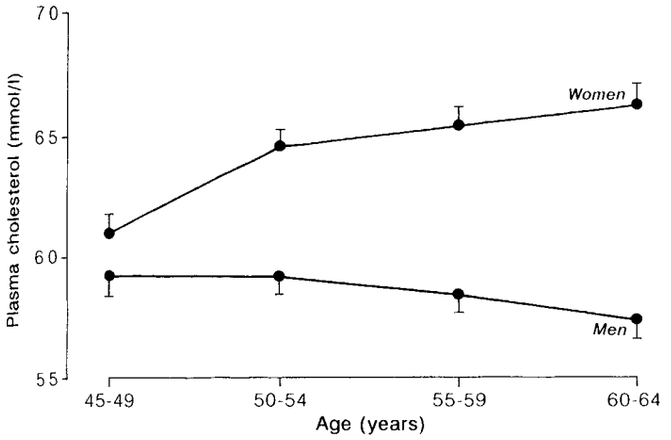

Fig 2—Mean (99% CI) plasma cholesterol in women and men.

Records kept with the Registrar General in Scotland ensured notification of a subject's death, provided it took place in the UK, together with the cause of death according to the 9th revision of the International Classification of Diseases (ICD) codes.[3] Although uncertainties about cause of death taken from the death certificate have been noted,[4] we had previously assessed the methods of the Registrar General in a study of hypertensive patients.[5] Notification from the Registrar General was virtually complete and coding comparisons of causes of death by the Registrar and by independent physicians yielded no important discrepancies. Full details of our methods have been published elsewhere.[6]

Our analysis concerns the relation between markers of coronary risk and coronary disease in women, and a comparison with better-known findings in men. For this study, we have taken 15-year mortality data based on 490 CHD deaths in women and 878 CHD deaths in men (ICD codes 410–414). CHD mortality rates were calculated as deaths per 1000 patient years of follow-up, and then adjusted for all variables (age, smoking, cholesterol, diastolic blood pressure, body mass index, and social class), except for the variable of interest, before the data were arranged in quintiles. The quintile points for cholesterol were 5·5, 6·1, 6·6, and 7·2 mmol/l in women, and 5·0, 5·6, 6·0, and 6·6 mmol/l in men. Corresponding values for diastolic blood pressure were 74, 80, 87, and 95 mm Hg in women and 74, 81, 88, and 96 mm Hg in men. Quintile points for body mass index were 22·2, 24·2, 26·2, and 28·9 kg/m² in women, and 23·0, 24·9, 26·6, and 28·5 kg/m² in men. CHD mortality was also assessed by smoking category (zero, 1–14, ⩾15 cigarettes per day, ex-smoker) and by social class (1 and 2, 3 non-manual, 3 manual, and 4 and 5). We applied a proportional hazards model to evaluate trends in mortality across each risk distribution after adjustment for other risk markers.

Results

Prevalence of risk markers in women

Fig 1 shows the distribution of plasma cholesterol in women and men. Mean (SD) values were higher in women (6·43 [1·09] mmol/l) than in men (5·87 [0·96] mmol/l). The proportion of women with cholesterol greater than 5·2, 6·5, and 7·8 mmol/l was 89·0%, 47·8%, and 11·2%, respectively; corresponding values for men were 77·6%, 26·3%, and 3·3%, respectively. The difference between the sexes was a result of higher cholesterol concentrations in older women than in older men (fig 2).

Women were less likely than men to smoke or to be heavy smokers (table I). Both average diastolic blood pressure and the proportion of subjects with a diastolic pressure greater than 90 mm Hg were similar in men and women. More men than women were overweight (body mass index 25–29 kg/m²) but a higher proportion of women were obese (body mass index >29 kg/m²).[7] Although husbands and wives shared social class, among those who were unmarried a higher proportion of women belonged to social classes 4 and 5 (table I).

TABLE I—DISTRIBUTION OF RISK MARKERS AMONG WOMEN AND MEN

—	Women (n=8262)	Men (n=7137)
No (%) subjects of smoking habit.		
Non-smoker	3824 *(45·8)*	1189 *(16·8)*
1–14 /day	1572 *(18·8)*	981 *(13·9)*
⩾15 /day	2334 *(28·0)*	3152 *(44·7)*
Ex-smoker	623 *(7·5)*	1736 *(24·6)*
Mean (SD) DBP (mm Hg)	85 (14)	86 (13)
No (%) subjects with DBP:		
<90 mm Hg	5537 *(66·3)*	4533 *(64·2)*
90–99 mm Hg	1631 *(19·5)*	1460 (20·7)
100–109 mm Hg	784 *(9·4)*	711 *(50·1)*
⩾110 mm Hg	397 *(4·8)*	350 *(5·0)*
Mean (SD) BMI (kg/m²)	25·8 (4·5)	25·9 (3·4)
No (%) subjects with BMI:*		
<18·5 kg/m²	190 *(2·3)*	53 *(0·8)*
18·5–25·0 kg/m²	3830 *(45·9)*	2813 *(39·9)*
25·0–29·0 kg/m²	2697 *(32·3)*	2995 *(42·5)*
⩾29·0 kg/m²	1662 *(19·5)*	1194 (16·9)
No (%) subjects of social class		
1+2	1455 *(17·4)*	1333 *(18·9)*
3 non-manual	1977 *(23·7)*	834 *(11·8)*
3 manual	1490 *(17·8)*	2816 *(39·9)*
4+5	3431 *(41·1)*	2075 *(29·4)*

DBP = diastolic blood pressure, BMI = body mass index.
* <18·5 is underweight, 18·5–25·0 ideal; 25·0–29·0 overweight, ⩾29·0 obese.[7]

Coronary risk and mortality

There were 490 CHD deaths in women and 878 CHD deaths in men. Although menopausal status was not known for individual patients, only one female coronary death occurred before age 52 (the average age of menopause in the UK). Thus, most mortalities among women in our study were postmenopausal.

15-year CHD mortality by baseline plasma cholesterol and other coronary risk markers is shown in figs 3 and 4. Plasma cholesterol, cigarette smoking, diastolic blood pressure, and low social class were positively and independently associated with CHD mortality in both sexes, whereas there was no relation between body mass index and CHD after adjustment for other risk markers.

The relative risk of coronary death (top to bottom quintile) was similar in both sexes (table II). In women, the absolute risk was less in that, for a given risk factor, women with high individual risks generally had a lower coronary mortality than men with low risks. Fig 3 shows that women with cholesterol concentrations above 7·2 mmol/l had a

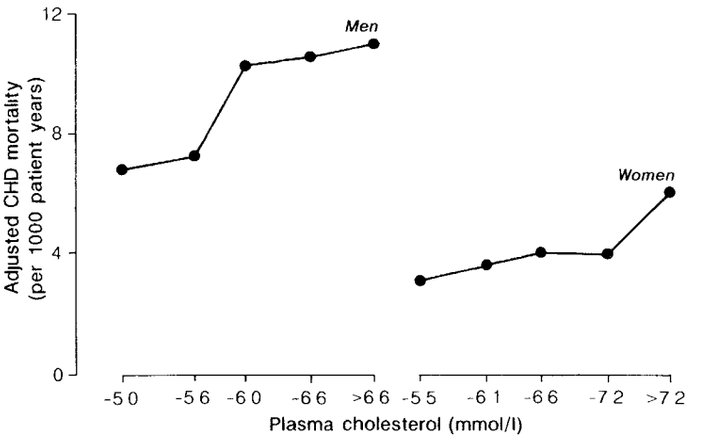

Fig 3—Adjusted CHD mortality in deaths per 1000 patient years for men and women by quintile of plasma cholesterol.

There were 490 CHD deaths in women with a relative risk of 1·77 ($p<0·001$). Corresponding figures for men were 878 CHD deaths, relative risk 1·56 ($p<0·001$).

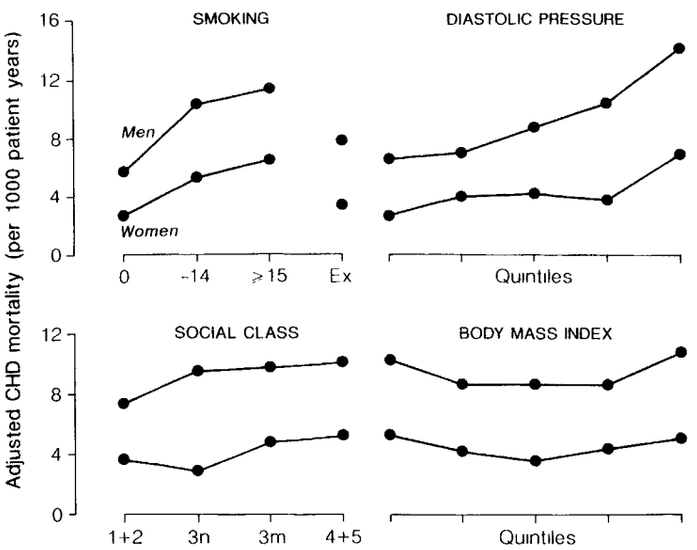

Fig 4—Adjusted CHD mortality in deaths per thousand patient years for both sexes by cigarette smoking, diastolic pressure, social class, and body mass index.

$p < 0.001$ for smoking, diastolic pressure, and social class No significant influence on CHD mortality was found for body mass index. For each of the four analyses risks were greater in men

lower risk of CHD than men with cholesterol values below 5·0 mmol/l. Similar trends were present for blood pressure, smoking, and social class (fig 4).

The best estimate of the number of CHD deaths attributable to any given risk marker is known as attributable risk—ie, the number of CHD deaths that would not have taken place if the levels of that risk marker had been in the lowest category of risk. All attributable risks in our population were less in women than in men (table II). More lives could have been saved if the whole population had not smoked than by any other single intervention, assuming that these other measures would be completely effective in reversing risk (table II). Calculation of attributable risk for cholesterol indicated that there would have been 103 (21%) fewer CHD deaths in women and 211 (24%) fewer CHD deaths in men if the mortality of the whole group had been the same as that among women and men whose cholesterol concentrations were in the lowest quintile. Attributable risks for diastolic blood pressure and low social class are also shown in table II.

TABLE II—RELATIVE AND ATTRIBUTABLE RISK OF CORONARY DEATH IN MEN AND WOMEN

—	Relative risk (95% CI)	p	Attributable risk (deaths saved)
Men			
Cholesterol	1·56 (1·32, 1·85)	<0·001	24% (211)
Smoking	2·03 (1·64, 2·52)	<0·001	40% (351)
Diastolic blood pressure	2·19 (1·74, 2·75)	<0·001	31% (272)
Body mass index	1·07 (0·87, 1·32)	NS	.. ..
Social class	1·45 (1·17, 1·80)	<0·001	25% (220)
Women			
Cholesterol	1·77 (1·45, 2·16)	<0·001	21% (103)
Smoking	2·38 (1·91, 2·96)	<0·001	37% (181)
Diastolic blood pressure	2·62 (1·92, 3·58)	<0·001	39% (191)
Body mass index	0·95 (0·72, 1·26)	NS	.. ..
Social class	1·51 (1·14, 1·99)	<0·01	32% (157)

NS, not-significant.
Relative risk for cholesterol, diastolic pressure, and body mass index is the ratio of the top to bottom quintile for each risk marker, for smokers it is the ratio of the rate for current smokers who smoke 15 or more cigarettes per day to the rate of lifelong non-smokers, for social class it is the rate for classes 4 and 5 combined to classes 1 and 2 combined

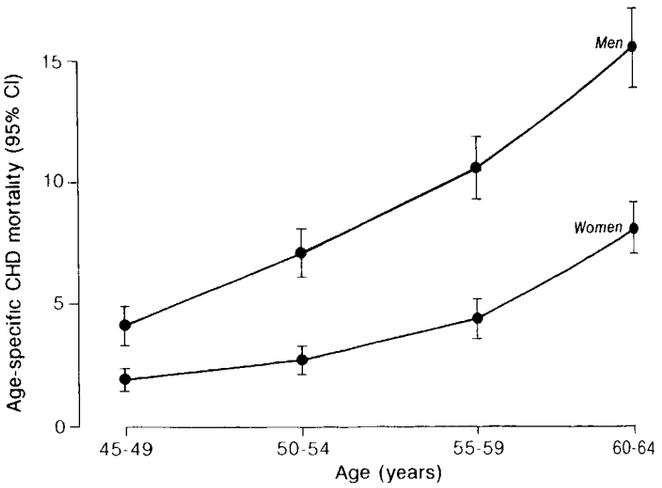

Fig 5—Age-specific CHD mortality (95% CI) adjusted for all other risk markers.

$p < 0.001$ for differences between sexes at all ages

The possibility that absolute risk was lower in women because women are protected by female sex hormones was investigated by calculating age-specific coronary death rates in both sexes after adjustment for all other risks (fig 5). Our results are consistent with this hypothesis and show that women have a substantially lower coronary mortality than men at every age studied. Although the gradient of risk tended to steepen in older women, postmenopausal women aged 60–64 years still had the same risk of coronary death as men 5–10 years younger and only half the coronary mortality of men aged 60–64 years.

Discussion

A recurring feature in most reviews about coronary prevention is the lack of data on women.[2] Few longitudinal studies on cholesterol[8–12] and only three of the major cholesterol-lowering drug trials[13–15] included women.

The Renfrew and Paisley survey is one of very few longitudinal health surveys in the UK to provide mortality data for women. The Scottish Heart Health Study[16,17] will give results for women in Scotland, but the screening survey has only recently been completed and it may be some time before mortality data are available. A case-control study of coronary risk factors among UK women has been published, but this did not include measurement of serum cholesterol.[18] One further advantage of the Renfrew and Paisley survey is the large number of coronary deaths in women (490), which is greater than the combined total of female coronary deaths (384) published in five other large surveys.[8–12]

The main limitations of our study are the lack of morbidity data and the absence of repeated measurements of risk markers during follow-up.[19] Death and its cause were certified by general practitioners but were not verified by independent observers. This method has disadvantages but is unlikely to introduce systematic bias.[19] Our approach has also been used by the Registrar General in his analysis of national statistics on CHD. Moreover, it is unlikely that cancer deaths will have been wrongly classified because the existence of a cancer registry in the west of Scotland allowed for independent confirmation.

Renfrew and Paisley have an especially high incidence of coronary disease. Our results indicate that the distribution of risk markers is different in men and women (table I, figs 1 and 2); that "dose responses" exist for cholesterol, cigarette smoking, diastolic blood pressure, and social class in both

sexes (figs 3 and 4); and that relative risks are similar in men and women (table II), although absolute and attributable risk are very much lower in women (table II, figs 3 and 4),

The reasons underlying the lower incidence of CHD in women remain unclear. Women may have a lower frequency of risk markers than men. Our survey suggests that they smoke less and have similar blood pressure, but have higher cholesterol and are more likely to be obese and of lower social class. However, the lower level of smoking among women cannot be the whole explanation for the difference in death rates because non-smoking women have only half the coronary mortality of non-smoking men (fig 4). Similar findings have been reported from other centres.[8] The social class differences between men and women are difficult to interpret because housewives and retired women assume their husbands' or fathers' social class. The large proportion of women who belong to social classes 4 and 5 is, however, likely to reflect unmarried women employed in textile, clothing, clerical, and service industries.

Women may tolerate risks better than men. We found that, in women, any given level of risk marker was associated with lower CHD rates than men. This protection might be conferred by female sex hormones. Our data are consistent with this explanation: even in women who were 60–64 years initially, CHD rates were substantially lower than in men of similar age, suggesting that natural oestrogen may protect for many years beyond the menopause (fig 5).

High-density lipoprotein (HDL) cholesterol was not measured routinely in our survey, although data from other centres[20] confirm that HDL is higher in women than in men and suggest that this difference may explain the lower female coronary rate. The relation between gender, lipoproteins, and coronary risk is complex, and a small sex difference in HDL concentration is unlikely to be entirely responsible for the large differences in CHD mortality that exist in Renfrew and Paisley. Non-contraceptive hormones in postmenopausal women may also confer protection against CHD,[20] but the frequency of hormone replacement therapy in Renfrew and Paisley was not recorded, although it is likely to have been low in the early 1970s. Triglyceride may have a more important influence on CHD risk in women than in men,[21] but it is unlikely that this could account for the sex differences observed in our study.

Several areas of uncertainty remain. Despite the lower incidence of CHD among women generally, diabetic women may have the same absolute risk of developing CHD as diabetic men.[8] Furthermore, women might be more likely than men to have angina or painless myocardial infarction as their initial presentation[8] and there are no sex differences in prognosis after myocardial infarction.[22]

How should women be advised? They should be urged not to smoke and to stop if they do smoke.[23] The evidence that control of blood pressure reduces the risk of myocardial infarction is weaker for women than it is for men,[24] but treatment of hypertension should be recommended because it is of proven value in the reduction of stroke in both sexes.[25] Regular physical exercise may protect against CHD, although such advice is derived from studies in men.[26]

Although body mass index was not an independent risk factor for CHD in our study, advice that obese women should lose weight is justified on general health grounds, and because weight loss is likely to facilitate management of hypertension and hyperlipidaemia. Indices of central obesity, such as waist-to-hip ratio and subscapular skin fold, may have greater predictive power than body mass index for CHD,[27] but neither were measured in Renfrew and Paisley.

Only three lipid-lowering studies have included women.[13-15] The first was a study of patients with pre-existing heart disease[13] and the second was of patients with familial hypercholesterolaemia.[14] Both studies suggested a benefit to women, but neither addressed the issue of primary prevention of CHD in women with moderately raised increases of their serum cholesterol. A third study was a secondary prevention trial in which the effects of cholestyramine and placebo on atheroma progression were compared; its results were inconclusive.

Definitive guidelines for the treatment of hypercholesterolaemia in women must await the results of further clinical trials. These will not be available for several years and until then any recommendations for women will have to be judged from estimates of risk rather than of benefit from reduction of risk. If absolute or attributable risk is the criterion on which such judgments should be based, the results of our survey suggest that for primary prevention of CHD the threshold for introduction of cholesterol-lowering drug therapy should be higher in women than in men.

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SHORT REPORTS

Glomerulonephritis with end-stage liver disease in childhood

G. NOBLE-JAMIESON S. THIRU
P. JOHNSTON P. FRIEND N. D. BARNES

Renal biopsies were done perioperatively in 18 children receiving liver grafts. All specimens showed glomerulonephritis, which was mesangial-proliferative in 15 and mesangio-capillary in 3. Of the 16 children who were alive four or more months after transplantation, only 1 showed progressive deterioration of renal function; 1 other had a subnormal but static glomerular filtration rate. In all 6 children who had proteinuria before operation, the urine became normal.

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The association of nephropathy with chronic liver disease has been thought infrequent.[1] Now that transplantation has become an accepted treatment for end-stage liver disease the question assumes greater importance: glomerulonephritis might complicate the postoperative course and worsen the long-term outcome. In 5 children with liver disease due to alpha-1-antitrypsin deficiency, evidence of renal dysfunction led us to do renal biopsies, and in every case the kidney showed abnormalities. We therefore investigated the association systematically in a further 13 children receiving first liver grafts.

The study was approved by an ethics committee and no parents withheld consent. This report includes the original 5 children with alpha-1-antitrypsin deficiency. The 13 children in the prospective series had liver disease of various causation (see table), and renal biopsy was done during liver grafting. These patients were selected consecutively unless at operation the procedure was judged dangerous (there were no complications). The specimens were divided and processed for routine histological examination, frozen-section immunofluorescence, and electronmicroscopy.

All 18 biopsy specimens showed abnormalities (see table): 15 children had a mesangialproliferative glomerulonephritis (immune staining positive for IgA in 8 and for IgM in 1; diffuse immune complex staining in 1). 3 children had mesangio-capillary glomerulonephritis. Tubular changes (mild necrosis) were seen in only 1 patient, who had potassium wasting disease.

16 children survived to follow-up at 4–34 months (mean 5 months). The urine became normal in all 7 who had shown proteinuria or haematuria before operation (proteinuria 3, proteinuria/haematuria 3, haematuria 1). Only 1 child showed clear long-term deterioration in renal function postoperatively: she had mesangio-capillary glomerulonephritis, and her glomerular filtration rate declined from normal to 25% in the year after transplantation. Another child, who had mesangialproliferative glomerulonephritis and a scarred right kidney, had a subnormal (25%) but stable glomerular filtration rate.

Blood-pressures tended to rise postoperatively (table), and 7 children had seizures or encephalopathy within the

PATIENT DETAILS

Patient	Diagnosis	Age (yr)	Renal biopsy		Highest mean arterial pressure (SD score)		Central nervous system complications
			Histology	Immune staining	Preop	Postop	
1*	Antitrypsin deficiency	14·5	MPGN	Not done	93 (+1·0)	130 (+4·6)	Encephalopathy (day 4)
2*	Antitrypsin deficiency	8·2	MPGN	Negative	102 (+2·8)	145 (+7·3)	Seizure/encephalopathy (day 6)
3*	Antitrypsin deficiency	3·8	MPGN	IgA	93 (+2·2)	126 (+5·4)	Encephalopathy (day 7)
4*	Antitrypsin deficiency	5·6	MCGN	N/A	86 (+1·5)	125 (+5·4)	Seizure/encephalopathy (day 26)
5*	Antitrypsin deficiency	8·5	MCGN	N/A	93 (+2·0)	114 (+4·0)	Nil
6	Biliary atresia	3·4	MPGN	Immune complex	87 (+1·8)	104 (+3·3)	Nil
7	Biliary atresia	12·2	MCGN	N/A	107 (+2·5)	135 (+5·1)	Nil
8	Familial cirrhosis	6·2	MPGN	IgA	72 (0·0)	112 (+4·1)	Seizure (day 21)
9	Biliary atresia	1·6	MPGN	Negative	93 (+2·0)	82 (+1·3)	Nil
10	Dicarboxylic aciduria	3·4	MPGN	IgA	70 (0·0)	101 (+3·1)	Nil
11	Alagille syndrome	13·8	MPGN	Not done	93 (+0·8)	90 (+0·8)	Nil
12	Biliary atresia	1·9	MPGN	IgA	70 (0·0)	83 (+1·4)	Nil
13	Antitrypsin deficiency	0·9	MPGN	IgA	77 (+1·0)	101 (+3·3)	Nil
14	Tyrosinaemia	3·9	MPGN	IgM	82 (+1·0)	110 (+3·9)	Nil
15	Biliary atresia	8·4	MPGN	Not done	65 (−1·0)	112 (+3·8)	Seizure (day 23)
16	Biliary atresia	11·1	MPGN	IgA	92 (+1·5)	103 (+2·4)	Seizure (day 13)
17	Biliary atresia	13·8	MPGN	IgA	80 (0·0)	97 (+1·3)	Nil
18	Biliary atresia	1·8	MPGN	IgA	95 (+2·5)	113 (+4·3)	Nil

*Patients studied before prospective series. MPGN = mesangialproliferative glomerulonephritis MCGN = mesangio-capillary glomerulonephritis. N/A = not applicable